982-117 Electroanatomical Validation of a New Non-Fluoroscopic Mapping System in the Swine Heart

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A major limitation in the currently used mapping methods is the inability to associate relevant electrophysiologic information with the endocardial locations from which they were acquired. Recently, a new non-fluoroscopic mapping (NFM) system was developed to solve this problem by creating real time 3-D electroanatomical maps of the heart chambers, allowing the correlation between the hearts electrical activation patterns and its anatomy. The purpose of this study was to validate the maps acquired by the NFM system with the same anatomical specimens studied postmortaly. Five healthy pigs were studied. The NFM system uses magnetic technology to accurately determine the location and orientation of the tip of a special catheter. By sampling a plurality of sites in which the tip was in stable contact with the endocardium the 3-D arrangement of the cardiac chamber is reconstructed in real time. The activation pattern is then color coded and superimposed on the reconstructed image. The electroanatomical maps obtained showed excellent correlation with the shape and morphological relationship between different anatomical structures for both the atria and ventricles in all hearts and in an additional endocardial cast. Similarly, we found high correlation between the locations of the conductive tissues verified histologicaly and the activation sequences produced by the catheter.

Conclusions: Our experience show excellent correlation between electranatomical maps obtained with the new NFM system and the gross and microscopic anatomy of the swines heart.

982-118 Local Capture by Atrial Pacing of Chronic Atrial Fibrillation in Human Beings

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The possibility of local atrial capture has been recently shown in experimental or induced AF in human beings. This study was undertaken to evaluate the feasibility of atrial capture (suggestive of an excitable gap) in spontaneous chronic AF. Decremental pacing was performed in 47 right atrial sites in 14 patients with chronic AF, not taking antiarrhythmic drugs. A Franz catheter (for pacing and monophasic action potential recording) and a recording quadripolar catheter positioned about 10 mm apart were used. Local capture was achieved in 41 (87%) sites for a total of 100 captures. In 71 episodes the capture was lost within 15 sec, while in the remaining 29 pacing was stopped after 15 sec of stable capture. The AF types immediately before capture were type 1 in 83 and type 2 in 17 episodes. Type 3 AF was never captured. Pacing cycle at capture was 175.7 ± 20.9 ms. The baseline FF interval was 185.4 ± 24.5, significantly longer than the FF recorded during pacing immediately before capture (176 ± 19.8 ms) ($p < 0.02$).

Conclusions: during spontaneous chronic AF in human beings: 1) local capture by atrial pacing is possible up to at least 15 mm from the pacing site; 2) regional entrainment is possible during type 1 and type 2 AF, but not in type 3 AF; 3) pacing before capture accelerates AF, probably by transient or very local capture. These findings suggest that an excitable gap is present in chronic AF, therefore supporting the hypothesis that leading circle re-entry or random re-entry are not the unique electrophysiological mechanisms maintaining the arrhythmia.

982-119 Evidence of Triggered Activity as the Mechanism of Atrial Tachycardia in Dogs With Pacing-induced Heart Failure

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We hypothesized that the mechanism of atrial tachycardia (AT) in dogs with ventricular pacing-induced heart failure (CHF) is triggered activity related to delayed after depolarizations (DAD). Conscious dogs (n = 8) were studied with 3 transvenous, chronic atrial leads at baseline and in CHF after 3–7 weeks of right ventricular pacing (200–250 bpm). AT inducibility, termination and pharmacologic responses were examined. In CHF, atrial refractory periods prolonged (116 ± 11 to 136 ± 15 ms, $p < 0.001$) without changes in dispersion or conduction times. At baseline, AT was induced in 0 dogs and in CHF, sustained (>30 min) AT was reproducibly induced in 8/8 dogs. AT (119 ± 9 ms) was induced in 8 dogs by burst pacing and in 5 dogs by extrastimuli. AT was more easily induced with shorter burst cycle lengths (CL) or extrastimuli and longer burst trains. The initial AT CL was directly related to the burst CL. Overdrive pacing accelerated AT in 8/8 dogs and terminated AT in 6/8 dogs. The burst CL required to terminate was 14 ± 8 ms < the AT CL. Terminations post pacing were not abrupt but associated with >10 beats of variable CLs before sinus rhythm. Resetting with single extrastimuli and entrainment could not be shown. Verapamil (2.5 ± 1.0 mg) terminated AT in 7/8 dogs after 4.6 ± 3.7 min and made reinduction more difficult. Flunarizine (56 ± 24 mg) terminated AT in 4/4 dogs after 3.2 ± 3.3 min and prevented reinduction. Lidocaine (69 ± 31 mg) terminated AT in 3/7 dogs and prolonged AT CL (+21 ± 9 ms). Adenosine (24 mg) converted AT to atrial fibrillation in 6/6 dogs. *Conclusion:* In this CHF model, responses to pacing and drugs suggest an AT mechanism due to DAD-induced triggered activity related to calcium overload.

982-120 Functional Mechanisms Underlying Tachycardia-induced Sustained Atrial Fibrillation in a Chronic Dog Model

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Rapid atrial activation is known to promote sustained atrial fibrillation (AF) ("AF begets AF"), but the underlying mechanisms are poorly understood. In order to assess the functional mechanisms by which rapid activation leads to development and maintenance of AF, we studied dogs with transvenous atrial pacing at 400 beats per minute for 1 (P1, n = 7), 7 (P7, n = 7) or 42 (P42, n = 3) days, and compared them with sham dogs (P0, n = 15) similarly instrumented but without pacemaker activation. Atrial pacing led to progressive increases in the duration of AF (dAF) induced by 15 trials of burst pacing. Epicardial mapping with a 112-electrode atrial array showed multiple reentry circuits (RC) during AF, the number of which increased with increased duration of rapid atrial pacing. Mean (± SE) functional changes were:

Gr	dAF(s)	RC	AF CL (ms)	CV (cm/s)	ERP (ms)	WL (cm)
P0	3.2 ± 0.3	1.5 ± 0.3	106 ± 2	108 ± 5	93 ± 3	10 ± 1
P1	40.3 ± 28.6	2.3 ± 0.4	102 ± 6	97 ± 8	95 ± 4	9 ± 1
P7	124.4 ± 49.7*	2.8 ± 0.3**	94 ± 4*	95 ± 8	91 ± 6	8 ± 1
P42	> 1800.0**	4.2 ± 0.2**	75 ± 7*	87 ± 1**	74 ± 8*	6 ± 1**

(AF CL = AF cycle length; CV = conduction velocity; ERP = effective refractory period; WL = wavelength; *$p < 0.05$, **$p < 0.01$ vs P0).

A progressive impairment in ERP accomodation to CL occurred over time, as previously described. *Conclusion:* Rapid atrial activation promotes the development of sustained AF by leading to progressive decreases in ERP and CV at short CL. These decrease the WL, allowing for a larger number of simultaneous RC that stabilize AF.

982-121 New Insights Regarding the Atrial Flutter Reentrant Circuit in the Canine Sterile Pericarditis Model

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We now have the ability to record simultaneously from the epicardium of both atria and the atrial septum using 388 electrodes in the sterile pericarditis atrial flutter (AFl) model. This permitted us to reexamine atrial activation during induced AFl to test the hypothesis that the AFl reentrant circuit includes a septal component. We studied 8 episodes of induced (rapid pacing or programmed stimulation), sustained (>5 min) AFl in 8 dogs. The mean AFl cycle length was 151 ± 13 ms (range: 134–170 ms), with a mean beat-to-beat oscillation of 2.3 ± 1.7 ms (range: 0–6 ms). In all 8 AFl episodes, one reentrant circuit included a septal component. In 4 episodes, there was a second reentrant circuit in the right atrial (RA) free wall. In those 4 episodes, the direction of the RA free wall circuit was clockwise in 2, and counterclockwise in 2. In the clockwise AFl, the septal wave front broke through to the epicardium at Bachmann's bundle and traveled behind the superior vena cava to enter the RA free wall. There it joined the RA free wall reentrant circuit (figure of eight reentry), travelling craniocaudally and reentering the atrial septum in the region of the inferior vena cava to complete the reentry. In the counterclockwise AFl, again figure of eight reentry was present, but the septal/RA free wall component travelled in the reverse direction compared to clockwise AFl. In the other 4 episodes, the only reentrant circuit included a septal component. In 3 of the 4 episodes, the activation wave front travelled caudocranially in the septum and craniocaudally in the area of the pectinate muscle in the RA free wall. It travelled the reverse direction in the fourth episode. Even in the absence of a RA free wall circuit, these 4 episodes exhibited a line of functional block in the area of the sulcus terminalis. *Conclusion:* In this model of AFl, reentry always 1) included a septal component; 2) did not require a RA free wall reentrant circuit; 3) demonstrated figure of eight reentry when

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